Loss of inflammatory signaling molecule protects mice against diet-induced obesity

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Obesity and subsequent complications are increasing in frequency worldwide. The accumulation of adipose tissue is associated with increased inflammation, and it has been proposed that modification of proinflammatory responses could alter adipose tissue composition.

A study in this issue of *JCI Insight* demonstrates that mice lacking the proinflammatory signaling molecule TAK1 are protected from obesity and insulin resistance induced by a high fat diet.

A team led by Antonia Sassmann-Schweda of the Max-Planck-Institute for Heart and Lung Research and colleagues generated mice lacking TAK1 specifically in adipocytes.

Compared to control animals, TAK1-deficient mice had reduced numbers of adipocytes and exhibited increased energy expenditure. Moreover, removal of TAK1 from mice that had previously been fed a high fat diet prevented additional weight gain and improved glucose tolerance.

The results of this study suggest that TAK1 should be further explored as a target for obesity treatment.

More information: Antonia Sassmann-Schweda et al, Increased apoptosis and browning of TAK1-deficient adipocytes protects against obesity, *JCI Insight* (2016). DOI: 10.1172/jci.insight.81175

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